From the Society for Vascular Surgery

A multicenter clinical trial of endovascular stent graft repair of acute catastrophes of the descending thoracic aorta

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Objective: **Thoracic endovascular aortic repair (TEVAR) is applicable to a spectrum of thoracic aortic pathology with half of the procedures performed world-wide for indications other than degenerative aneurysm of the descending thoracic aorta (DTA). This multicenter, prospective study queried perioperative and one-year results of TEVAR using the commercially available GORE TAG device, in the treatment of acute complicated Type B dissection (cTBD), traumatic aortic tear (TT), and ruptured degenerative aneurysm (RDA) of the DTA.**

Methods: **This prospective, non-randomized, literature controlled study included 59 patients; cTBD, n = 19; RDA, n = 20; TT, n = 20. The primary end-point was the composite of death and total paraplegia in subjects at ≤ 30 days post-treatment compared with a cohort from current literature. Secondary end-points included adverse events related to device, procedural and systemic complications, and one-year survival.**

Results: **All 59 patients had successful endoprosthesis deployment. Fifteen of 19 (79%) patients in the cTBD group had either rupture or malperfusion syndromes at presentation. Combined 30-day mortality/paraplegia rate was 13.6% (8/59), with seven (11.9%) deaths (cTBD [3], RDA [3] and TT [1]) and 1 (TT, 1.7%) case of paraplegia. The primary end-point for the TEVAR cohort was significantly lower ($P = .008$) when compared with a composite literature control of 800 patients (combined 30-day mortality/paraplegia of 29.6%). Thirty-day complications of any nature occurred in 48 (81%) patients; 11 (18.6%) were device related, and 43 (73%) experienced one or more systemic adverse events. Six (10%) patients required additional TEVAR implantations and 3 (5%) patients (one in each pathology group) required conversion to open surgery. Seventeen (29%) patients had endoleaks of any kind or degree through 30 days; cTBD (7), TT (2), RDA (8). Nine patients (15.3%) had perioperative strokes with two resultant deaths. During mean follow-up time of 409 ± 309 days, an additional 12 patients died, one patient required open conversion (cTBD), and two patients had major device related events. Actuarial survival at one year was 66% (range, 52%-77%) for the entire cohort; (cTBD) 79% (range, 53%-92%), (TT) 79% (range, 53%-92%) and (RDA) 37% (range, 16%-59%). On regression analysis, age at treatment (1.05 [range, 1.01-1.09]; $P = .008$) and chronic obstructive pulmonary disease (COPD) (4.3 [range, 1.3-14.4]; $P = .02$) were predictive of death at one year.**

Conclusion: **This study confirmed treatment advantages for TEVAR for thoracic aortic catastrophes when compared with literature-based results of open repair. One-year treatment results indicate a low incidence of graft-related complications. TEVAR is the preferred initial treatment for the DTA catastrophes studied herein. (J Vasc Surg 2009;50:1255-64.)**

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Table I. Inclusion/exclusion criteria for TAG Complex Pathology Trial

General inclusion criteria
Aneurysmal rupture, aortobronchial/aortoesophageal fistula of the DTA, *or Traumatic transection* of the DTA, *or Acute complicated distal aortic dissection*, *and* Subject must be ≥ 18 years old, able to comply with consent and follow-up and deemed a candidate for open surgical repair by the investigator, and Arterial anatomy meets pre-defined criteria (seal zone arterial diameters and lengths as per original TAG device FDA approval)
Disease-specific inclusion criteria
Aneurysmal rupture, aortobronchial fistula, aortoesophageal fistula
Ruptured thoracic aneurysm, or thoracic aortobronchial or aortoesphageal fistula
Traumatic transection
Complete or incomplete transection of the DTA caused by a traumatic event
Acute complicated distal aortic dissection
Subject diagnosed within 14 days of symptom onset, *and*
Complicated course, which must include one of the following:
End organ ischemia (ie, malperfusion syndrome)
Refractory hypertension
Rupture or impending rupture
Uncontrollable pain, and
Entire dissection is distal to the left subclavian artery (ie, retrograde dissection from the distal thoracic aorta is an exclusion criterion)
Exclusion criteria
4 mm diameter taper between proximal and distal landing zones of DTA and inability to use devices of different diameters to compensate for the taper
Not an exclusion criterion for aortic dissection cases. Endoprosthesis to be implanted within true lumen.
Marked tortuosity or stenosis of iliac and/or femoral arteries
Significant thrombus at the proximal or distal landing zones
Mycotic aneurysms
Paraplegia or paraparesis at initial presentation
Participation in another drug or medical device study within one year of study enrollment

DTA, Descending thoracic aorta.

Comparative clinical trials support the preferential use of thoracic endovascular aortic repair (TEVAR; vs. open surgery) for treatment of intact degenerative aneurysms of the descending thoracic aorta (DTA).[1-4] Yet, unlike endovascular aortic repair (EVAR) for treatment of degenerative abdominal aortic aneurysm (AAA), TEVAR is potentially applicable to the spectrum of thoracic aortic pathology. Indeed, world-wide implants of the sponsor's "TAG" endograft (GORE TAG Thoracic Endoprosthesis, W.L. Gore, Inc., Flagstaff, Arizona, USA) include some 45% of procedures performed for pathologies other than degenerative aneurysm of the DTA.[5] Despite the fact that device engineering considerations might differ among different pathologies, TEVAR has gained wide acceptance among surgeons treating, in particular, acute potentially catastrophic conditions of the DTA.[6-11] Following initial FDA approval of the TAG device for treatment of intact degenerative aneurysm of the DTA in March 2005, a multicenter clinical trial was designed to test its efficacy in the challenging DTA pathologies of ruptured degenerative aneurysm (RDA), traumatic tear (TT), and acute complicated Type B aortic dissections (cTBD). The trial was conducted at 14 U.S. academic centers (Appendix A, online only); enrollment was completed in February 2007, and results with one-year follow-up are reported herein.

METHODS

This study is a prospective, non-randomized, literature-controlled, multicenter trial, which took place between August 2005 and February 2007. Enrollment was open to subjects with the following conditions: (1) RDA of the DTA, including aortobronchial or aortoesophageal fistula; (2) TT of the DTA; and (3) cTBD of the DTA as defined in Table I, which also details overall inclusion/exclusion criteria. All subjects were deemed candidates for open repair for comparison to open surgical literature controls. Investigators were allowed to enroll any patient within the anatomic inclusion criteria. Eligible subjects underwent pre-treatment extremity neurologic evaluation, non-contrast and contrast-enhanced spiral computed tomography (CT) with multiplanar reconstruction. Enrollment was made once patients signed informed consent. A literature search revealed projected major advantages for endovascular repairs as detailed in Appendix B (online only). We conducted a thorough review of peer-reviewed literature to estimate the incidence of death and paraplegia through 30 days post-treatment with open repair for complex pathology of the DTA. Manuscripts published in the 15 years prior to study initiation (1989-2004) were selected. We included up to eight manuscripts (19 total) per pathology, each with a minimum total sample size of 50 subjects per pathology (800 total). The primary endpoint estimates calculated from these manuscripts were summarized and presented to a panel of cardiothoracic and vascular surgeons, which corroborated the results of the analysis. The protocol was both FDA and individual centers' institutional review board-approved with literature controls, indicating open repair had a 30-day estimated 29.6% mortality/paraplegia risk; the corresponding figure for TEVAR was 12.6%. Based on pre-study power analysis, it was estimated that 52 test subjects with primary endpoint data would be required to detect a difference of 17% in the composite outcome; 20 subjects were enrolled in

Table II. Clinical inclusion criteria for patients in the complicated Type B dissection group

Pt.	*Malperfusion*	*Organ involved*	*Presenting symptoms of ischemia*	*Refractory HTN*	*Rupture*	*Pain**	*Number of symptoms*
1	No			Yes	No	Yes	2
2	No			Yes	No	Yes	2
3	Yes	Kidney	Increased creatinine	No	Yes	No	2
4	No			No	Yes	Yes	2
5	Yes	Extremity	Decreased pulse and cyanosis	No	No	No	1
6	No			Yes	No	No	1
7	Yes	Bilateral extremity	Absent & diminished femoral pulses	No	No	Yes	2
8	No			No	No	Yes	1
9	No			No	Yes	No	1
10	Yes	Kidney and extremity	Chest and back pain	No	No	No	1
11	Yes	Extremity	Leg ischemia	No	No	No	1
12	No			No	Yes	No	1
13	No			No	Yes	No	1
14	No			No	Yes	No	1
15	No			No	Yes	No	1
16	No			Yes	Yes	Yes	3
17	No			Yes	Yes	Yes	3
18	Yes	Kidney and mesenteric	Extremity ischemia	Yes	No	Yes	3
19	Yes	Kidney and extremity	Increased creatinine	No	No	No	1

HTN, Hypertension.
*Despite medical treatment.

each arm. Primary end-point was the 30-day composite of death and total paraplegia. Lower extremity motor function post anesthesia, at hospital discharge and at 30 days was assessed using a standardized scale.[12] Secondary end-points included adverse events, device efficacy, one year survival, and graft performance data.

Follow-up protocol. Follow-up visits at 30 days, six months, and annually for five years post-treatment included clinical and CT exams. Clinical data were reported by individual centers and monitored independently; the sponsor's core laboratory collected CT scan data. A clinical events committee (CEC) was assembled to ensure accurate and consistent reporting of all adverse events (AE), and was responsible for final adjudication of the data. Adverse events were classified as major or minor according to previously defined reporting standards.[13] Actuarial survival is reported with Kaplan-Meier curves. A univariate model was used to analyze variables associated with death through the one-year period. Results are reported as [Odds Ratio (95% confidence interval); *P* value]. Variables with $P \leq .1$ were then entered into a multivariate Cox regression model and significant ($P \leq .05$) results are reported. χ^2 test was used to compare nominal data; $P < .05$ was considered significant.

The TAG thoracic endoprosthesis used in this trial has been previously described.[4] Only the surgeon investigators (ie, not sponsors' clinical study staff) had access to pre-procedural imaging. Anatomic inclusion criteria for all pathologies mandated a proximal aortic seal zone between 23 mm and 37 mm and >2 cm in length. Such proximal seal zone could involve coverage of the left subclavian artery at the operators' discretion (but not the common carotid or innominate arteries) and by protocol had to be free of dissection or tear. Similarly, distal seal zones of 23 mm to 37 mm diameter and 2 cm in length (not relevant to dissection cases) were protocol mandated.

RESULTS

Demographics and clinical features. A total of 59 patients were enrolled; 19 in the cTBD group (one treated patient was post-hoc reassigned to the RDA group), 20 in the TT group, and 20 in the RDA group. As noted in Table II, clinical inclusion criteria in the dissection group included patients with renal (4), lower extremity (5), and/or mesenteric malperfusion syndromes (1). In total, 15/19 (78%) dissection patients were treated for rupture (9) and/or malperfusion (7), one of these patients having evidence of both.

Demographic and clinical features are displayed in Table III. Forty-seven (80%) patients were male. Patients in the RDA group were significantly ($P = .0004$) older than those in the cTBD and TT groups. Patients in the RDA group had significantly higher prevalence of coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), hypertension (HTN), and smoking than the TT group. Summary SVS (Society of Vascular Surgery) risk score for the entire cohort was 6.2 ± 6.1 (range, 0-24), with significantly higher values noted for the RDA cohort, and lower values noted for the TT cohort. For the TT group, mean injury severity score (ISS) was 36.1 ± 19.9 (range, 1-75).

Operative procedure and reinterventions. All 59 patients had successful deployment of the endoprosthesis. Thirty-eight (64%), 14 (24%), and seven (12%) patients had one, two, and three endoprosthesis placed, respectively, at initial procedure. On average, patients required 1.7 (range, 1-3), 1.1 (range, 1-2), and 1.7 (range, 1-3) devices in the cTBD, TT, and RDA groups, respectively. Six subjects required additional endograft implantation as follows: (1) (RDA) had a type Ia endoleak on CT done on post treatment day (PTD) one repaired with a proximal extension and coil embolization of the left subclavian artery on PTD three; (2) (RDA) had a type Ia endoleak on CT performed

Table III. Demographics and clinical features

	Primary pathology				
	Acute complicated dissection n (%)	*Traumatic transection n (%)*	*Aneurysm rupture n (%)*	*Overall total N*	P *value*
Subjects enrolled	19	20	20	59	
Mean age (years)	58.9 ± 14.7	51.3 ± 21.8	76.2 ± 10.7	62.2 ± 19.3	.0004
Risk factors					
Coronary artery disease	0 (0.0%)	3 (15.0%)	7 (35.0%)	10 (16.9%)	.01
Hypercholesterolemia	3 (15.8%)	4 (20.0%)	9 (45.0%)	16 (27.1%)	.08
COPD	3 (15.8%)	0 (0.0%)	6 (30.0%)	9 (15.3%)	.03
Congestive heart failure	1 (5.3%)	1 (5.0%)	2 (10.0%)	4 (6.8%)	.78
Hypertension	18 (94.7%)	8 (40.0%)	18 (90.0%)	44 (74.6%)	<.0001
Cigarette smoking	8 (42.1%)	5 (25.0%)	13 (65.0%)	26 (44.1%)	.04
Renal insufficiency	3 (15.8%)	1 (5.0%)	3 (15.0%)	7 (11.9%)	.50
Stroke	3 (15.8%)	0 (0.0%)	2 (10.0%)	5 (8.5%)	.20
Diabetes	2 (10.5%)	4 (20.0%)	5 (25.0%)	11 (18.6%)	.50
Prior thoracotomy	1 (5.3%)	0 (0.0%)	4 (20.0%)	5 (8.5%)	.06
Summary SVS Risk Score (Mean, Std Dev)	6.1 (5.5)	4.8 (7.2)	7.6 (5.6)	6.2 (6.1)	.02

COPD, Chronic obstructive pulmonary disease; *SVS,* Society of Vascular Surgery.

Table IV. Primary endpoints through 30 days

	Primary pathology						
	Acute complicated dissection n (%)	*Traumatic transection n (%)*	*Aneurysm rupture n (%)*	*Overall total n*	*Open repair (literature controls)*	*Estimated risk difference (95% CI)*[1]	P *value*[2]
Subjects with successful delivery	19	20	20	59	800		
Mortality or paraplegia	3 (16%)	2 (10%)	3 (15%)	8 (13.6%)	237 (29.6%)	0.16 (0.07-0.25)	0.008
Mortality	3 (16%)	1 (5%)	3 (15%)	7 (12%)	193 (24.1%)	0.5 (0.24-1.0)	0.04
Paraplegia	0 (0%)	1 (5%)	0 (0%)	1 (1.7%)	44 (5.5%)	0.31 (0.04-2.2)	0.36

[1]Where risk difference is the proportion of control subjects - proportion of test subjects; (0.296-0.136 = 0.16).
[2]*P* values are based on Chi-square test of independent proportions.

on PTD two. This was treated unsuccessfully with a proximal extension and later successfully by embolization on PTD 98; (3) (RDA) had a type I endoleak found on CT on PTD-1 with unsuccessful proximal extension on PTD seven, a type Ia endoleak was still present on subject's death on PTD 121; (4) (TT) had an asymptomatic type I endoleak and proximal device compression found at six-month follow-up visit; proximal endograft extension initially failed but at one year no further device compression and endoleak resolution noted; (5) (cTBD) required a graft extension on PTD 27 for persistent filling of the false lumen distal to the graft. The patient remains stable at one-year follow-up, albeit the distal false lumen is still being perfused; (6) (cTBD) has had a complicated course that included open repair of an acute (separate event, not device related per imaging performed at the time of presentation) Type A dissection on PTD 40, a distal extension with a TAG device to extend the true lumen for superior mesenteric artery malperfusion and proximal extension to repair a proximal endoleak on PTD 91. On PTD 307, the patient presented with a contained rupture of the aortic arch which necessitated open conversion. Three patients (one in each group) required conversion to open surgery within 30 days, one because of rupture (cTBD), one due to an aorto-esophageal fistula (RDA), which was not observed on imaging done at the time of initial presentation, and the third due to device compression (TT).

Forty-eight (81%) patients had femoral cutdowns, while 11 (19%) patients had percutaneous access for delivery, the use of which was left at the discretion of individual site investigators. Procedure time averaged 108 ± 51 (range, 45-300) minutes. Procedure time was significantly shorter ($P < .05$) for the TT compared with the RDA group (85 ± 25 vs. 133 ± 68 minutes). Procedural blood loss averaged 301 ± 375 ml (range, 0-2000 ml). This was not significantly ($P = .6$) different between the three groups. Time in the intensive care unit averaged 9 ± 12 (range, 0.4-56) days. This was significantly greater ($P < .05$) for the TT group compared with other two groups (TT: 18.2 ± 16.7; RDA: 3.9 ± 3.9; cTBD: 4.6 ± 3.6 days). Hospital stay averaged 16 ± 24 (range, 1-149) days, which was significantly longer ($P < .05$) for the TT group compared with the other two groups (TT: 31 ± 36.6; RDA: 7.5 ± 5.1; cTBD: 9.1 ± 7.1).

Primary outcomes. Primary outcomes are detailed in Table IV. Combined 30-day mortality/paraplegia for the

Table V. Univariate analysis of variables associated with death at one year

Variable	*# Obs. included*	*Standard error*	P *value*	*Odds ratio*	*95% CI for OR*
Gender	59	0.495	0.183	1.932	(0.733, 5.095)
Age (years)*	59	0.019	<.001	1.065	(1.027, 1.105)
Race (Caucasian/Other)	59	1.028	0.219	3.535	(0.472, 26.488)
SVS Summary (Total)	58	0.031	0.127	1.048	(0.987, 1.113)
Pathology group*	59	0.315	0.029	1.989	(1.073, 3.685)
Height (cm)	57	0.021	0.228	0.976	(0.937, 1.016)
Weight (kg)*	59	0.011	0.062	0.980	(0.959, 1.001)
Body surface area	57	0.835	0.102	0.256	(0.050, 1.313)
Smoking history	59	0.460	0.372	1.507	(0.612, 3.713)
ASA	59	0.330	0.174	1.567	(0.820, 2.995)
Number of devices used*	59	0.291	0.035	1.850	(1.045, 3.274)
Coronary artery disease*	59	0.481	0.008	3.591	(1.398, 9.220)
COPD*	59	0.495	0.006	3.954	(1.498, 10.439)
CHF*	59	0.633	0.090	2.925	(0.846, 10.114)
Renal insufficiency	59	0.563	0.271	1.859	(0.616, 5.608)
Diabetes mellitus	59	0.494	0.190	1.913	(0.726, 5.040)
Peripheral vascular disease*	59	0.523	0.065	2.619	(0.940, 7.297)
Back pain symptom	57	0.460	0.645	0.809	(0.329, 1.993)
Chest pain symptom*	57	0.476	0.013	0.307	(0.121, 0.781)
Abdominal pain symptom	57	0.563	0.365	0.600	(0.199, 1.811)
Hypotension symptom	58	0.748	0.958	0.962	(0.222, 4.163)
Procedure time	59	0.004	0.256	1.004	(0.997, 1.012)
Procedural blood loss	58	0.000	0.096	1.001	(1.000, 1.002)

ASA, American Society of Anesthesiology; *CHF,* congestive heart failure; *CI,* confidence interval; *COPD,* chronic obstructive pulmonary disease; *OR,* odds ratio; *SVS,* Society of Vascular Surgery.
*Included in final multivariate Cox model.

study group was 13.6% (8/59). Seven (11.9%) patients died, and one (1.7%) patient (TT group) became paraplegic, the single spinal cord complication in the study. The combined mortality/paraplegia rate for the TEVAR cohort was significantly less ($P = .008$) when compared with the composite literature control of 800 patients (Table IV). Of the seven early deaths, three occurred in the cTBD group (two strokes, one aortic rupture), three in the RDA group (sepsis, stroke, myocardial infarction [MI]) and one in the TT group (acute respiratory distress syndrome [ARDS] secondary to pulmonary contusion). Univariate analysis for variables associated with death at one year is shown in Table V. Multivariate Cox regression analysis revealed age [1.05 (95% CI, 1.01-1.09); $P = .008$] and COPD [4.3 (95% CI, 1.3-14.4); $P = .02$] were predictive of death at one year.

Early adverse events. Major adverse events within 30 days occurred in 48 (81%) patients and are detailed in Table VI. Eleven (18.6%) of these were classified as device-related events, seven subjects (12%) experienced other procedure related events, and 43 (73%) subjects experienced one or more systemic adverse events. Two aortic ruptures occurred in the cTBD group within 30 days. The first patient presented with chest and back pain on PTD 29. A CT showed suspected contained rupture, which was managed with explantation and open conversion, complicated by a fatal CVA. The second patient had a complicated operative course, failure to seal the primary tear, and expired from rupture on PTD one. Partial proximal endograft collapse occurred in two patients (TT); one was repaired with placement of a second endograft and one with open conversion. Two patients in the RDA group had other implant related complications. The first patient had a type Ia endoleak identified on PTD two requiring three additional procedures for resolution. The second patient initially had a ruptured pseudoaneurysm proximal to a previously placed TAG device done for TT five months earlier, and presented with an aortoesophageal fistula on PTD 28. Endoprosthesis extrusion was noted at explantation during open repair. Two cTBD patients had major device related events (endoleaks) that necessitated revisions. The first patient had persistent perfusion of the false lumen on PTD 69 despite a revision procedure done on PTD 40 after the initial implant. The second patient presented with chest and back pain on PTD 31 and a CT showed an acute Type A dissection (ie, new ascending aortic tear identified, not retrograde dissection from graft) that required open surgery with the most proximal TAG device as the distal end of the surgical reconstruction.

Endoleaks. Seventeen (29%) patients had endoleaks of any degree or nature through 30 days. Seven endoleaks occurred in the cTBD, two in the TT, and eight in the RDA group. Twelve (71%) of the endoleaks were classified as type I (nine were type Ia and three Ib), four (24%) type II, and one could not be classified. Of these, six endoleaks were classified as major events because they resulted in conversion, rupture, or death (Table VI). Two endoleaks occurred in the cTBD group (type Ia and type II), one in the TT group (type Ia), and three in the RDA group (all type Ia). Three patients, all in the RDA group, underwent revisions due to persistent endoleak during the early period as above.

Table VI. Early and late major adverse events for TAG Complex Pathology Trial

	Early events: < 30 days				*Late events*		
	Acute complicated dissection n (%)	*Traumatic transection n (%)*	*Aneurysm rupture n (%)*	*Total*	*6-month*	*12-month*	*Total*
Patients	19	20	20	59	47	35	
Subjects with one or more major adverse events	16 (84%)	16 (80%)	16 (80%)	**48 (81.4%)**	17 (36%)	9 (26%)	**26**
Subjects with one or more major device-related adverse events	4 (21%)	2 (10%)	5 (25%)	**11 (18.6%)**	4 (8.5%)		**4**
Ruptured aorta	2 (11%)			**2 (3.4%)**			
Endograft infection			1 (5%)	**1 (1.7%)**	1 (2.1%)		**1**
Graft collapse		2 (10%)		**2 (3.4%)**	1 (2.1%)		**1**
Branched vessel occlusion					2 (4.2%)		
Endoleak	2 (11%)	1 (5%)	3 (15%)	**6 (10.2%)**	1 (2.1%)		**1**
Other implant related complication			2 (10%)	**2 (3.4%)**			
Subjects with one or more major procedure-related adverse events	3 (16%)		4 (20%)	**7 (11.9%)**			
Operative bleeding	1 (5%)		2 (10%)	**3 (5.1%)**			
Aortic dissection (within 30 days of treatment)	2 (11%)			**2 (3.4%)**			
Arterial perforation or rupture			3 (15%)	**3 (5.1%)**			
Access site lymphocele, lymphorrhea, lymphedema			1 (5%)	**1 (1.7%)**			
Subjects with one or more major systemic adverse event	14 (74%)	15 (75%)	14 (70%)	**43 (72.9%)**	15 (32%)	9 (25.7%)	**26**
Cardiac	3 (16%)	2 (10%)	4 (20%)	**9 (15.3%)**	5 (11%)		**5**
Pulmonary	5 (26%)	11 (55%)	7 (35%)	**23 (39.0%)**	4 (9%)		**4**
Renal insufficiency	2 (11%)	3 (15%)	1 (5%)	**6 (10.2%)**		1 (2.9%)	**1**
Cerebrovascular	4 (21%)	4 (20%)	1 (5%)	**9 (15.3%)**	2 (4%)	1 (2.9%)	**3**
DVT/PE/coagulopathy	2 (10%)	6 (30%)	1 (5%)	**9 (15.3%)**	1 (2%)		**1**
Bowel ischemia			1 (5%)	**1 (1.7%)**		1 (2.9%)	**1**
Spinal cord ischemia		1 (5%)		**1 (1.7%)**			**1**
Other systemic complication	8 (42%)	8 (40%)	6 (30%)	**22 (37.3%)**	8 (17%)	7 (20%)	**15**

DVT, Deep vein thrombosis; *PE*, pulmonary embolism.

Neurologic complications. One patient (normal pretreatment lower extremity function) in the TT group suffered paraplegia. The endograft covered both the left subclavian and the left vertebral artery which arose directly from the aortic arch. A magnetic resonance imaging (MRI) on PTD two showed changes consistent with spinal cord infarction at the T4 level, consistent with the patient's physical examination (T4-5 motor and sensory deficits). Nine patients (15.3%) had strokes during the early study period. Two patients died within 30 days related to a stroke. The first patient (cTBD) was an 81-year-old whose care was withdrawn after a CT confirmed a middle cerebral artery stroke. The second patient did not regain consciousness postoperatively after implantation of three TAG devices for a ruptured aneurysm. Care was withdrawn after a CT showed multiple embolic infarcts.

Late events. Twenty-six of 37 (70%) eligible patients had protocol visits at one year. An additional 12 patients died at this time interval. A total of three additional patients have been withdrawn from the study; one in the TT and two in the RDA group. The patient in the TT group was removed due open conversion. Of the two patients in the RDA group that were removed, the first declined appropriate follow-up in the study, and the second was removed for medical co-morbidities precluding follow-up. Conversion to open repair occurred in one patient in the cTBD group. This patient with a late acute Type A dissection was detailed above. One late, ultimately fatal endograft infection (RDA) occurred. An endoleak, related to partial graft collapse, was identified in one patient (TT) on late follow-up (PTD 87). This was successfully treated with a proximal endograft extension. Two additional patients had major device related events secondary to true/false lumen changes caudal to the stented segment during late follow-up. The first (cTBD) had superior mesenteric artery malperfusion, which required an additional TAG device to further extend the true lumen. In total there were four late graft events, two in the same patient. Kaplan-Meier analysis of time to first device-related major adverse event through one year is presented in Fig 1, with actuarial tables presented in Appendix C (online only). Anatomic remodeling in patients treated for cTBD was favorable with true lumen expansion and decrement in overall aortic diameter. These data have been separately reported.[14] Fig 2 shows a Kaplan-Meier analysis of survival through one year and actuarial tables are shown Appendix D (online only). Actuarial survival was 66% (range, 52%-77%) for the entire cohort; 79% (range, 53%-92%), 79% (range, 53%-92%) and 37% (range, 16%-59%) for the cTBD, TT, and RDA groups respectively. A log-rank test was performed to compare the survival curves

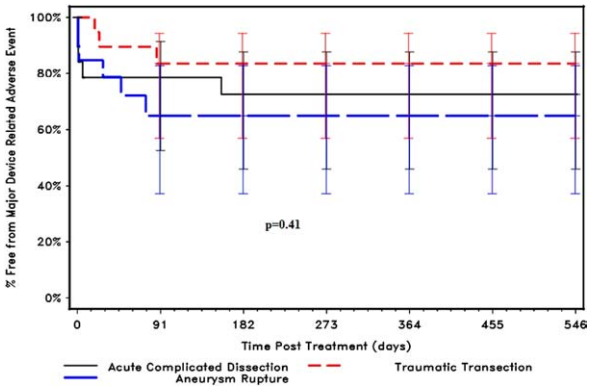

Fig 1. Kaplan-Meier analysis of time to first major device related adverse event.

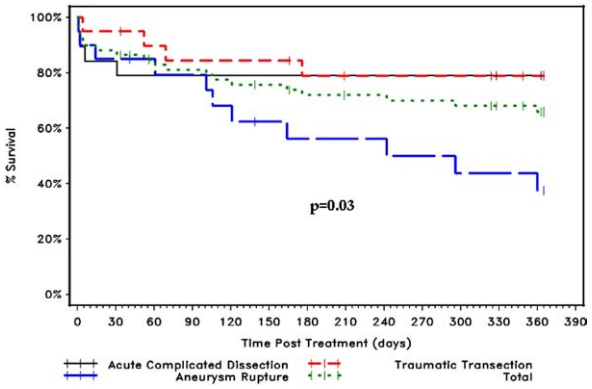

Fig 2. Kaplan-Meir analysis of survival for TAG Complex Pathology Trial.

through one year between the pathology groups; RDA subjects had shorter survival times than cTBD or TT ($P = .03$); however, when controlling for baseline co-morbidities (COPD, age), the effect of pathology is not significant. Two patients died on long term follow-up due to aortic related complications. At one year, freedom from aortic related death was 84.5% (95% confidence interval [CI]: 72.3-91.6).

DISCUSSION

As anticipated from review of the extant literature, periprocedural treatment morbidity was significantly in favor of TEVAR when compared with surgical literature controls. Both in the composite endpoint approved in the original protocol design, and in the individual pathologies (see below), treatment results with TEVAR compare favorably with conventional surgical treatment. For example, a 30-day mortality of 15% for treatment of ruptured DTA must be considered optimal when compared with corresponding figures of 28% and 27% from two centers of excellence.[15,16] The overall results are furthermore encouraging in the context of the relatively early evolution of TEVAR design constructs. Pathology-specific TEVAR constructs for complicated type B dissections[17] have been described and are currently being studied in multicenter clinical trials. The sponsor of the current study has modified the TAG device to preclude the problems with lesser curve malapposition and graft collapse; this device will soon be in clinical trials for both traumatic tear and cTBD.

The validity of historical controls is an anticipated criticism of single-arm clinical trials. In our study, this was borne of ethical considerations since the TAG investigators would (in current practice) preferentially treat the pathologies studied herein with a TEVAR strategy. Furthermore, the logical argument that "historical" controls do not reflect results potentially achievable (in this case with open surgery) in contemporary practice is refuted by both a review of current literature and our recalculation of the original point estimates based on the most recent available data. Examples include the current International Registry of Acute Aortic Dissection (IRAD) data for complicated type B acute dissection treated with TEVAR vs. open surgey.[18,19] A recent IRAD publication reveals a consistent 34% mortality associated with open surgical treatment of complicated type B dissections.[19] In large 2008 multicenter or meta-analysis publications referable to comparative TEVAR vs. open surgical repair of traumatic aortic tear, composite death/paraplegia for open surgical treatment was in the 21%-26% range.[7,20] Finally, a recent National Inpatient Sampling (NIS) study of open surgical treatment of degenerative DTA reported a "real world' mortality of 45% for RDA cases.[21] Accordingly, the recalculated point estimates for the historical literature controls reflect results anticipated in contemporary practice for these challenging pathologies treated with conventional surgery.

Our study included one-year follow-up data. Not surprisingly, given both the advanced age and the nature of the pathology in the RDA cohort, late survival was statistically inferior in this subgroup. Other studies have emphasized that 30-day outcomes underestimate the total impact of DTA disease and/or its treatment.[22] Survival in the 80% range at one year for TT and cTBD compares equally or favorably with the bulk of reported literature.[5-7,20,23] Alternatively, one-year survival of 37% in a cohort whose mean age was 76 years being treated for RDA emphasizes both respectable salvage in this subgroup, and the important reality of attrition in the first year after treatment. Rarely was this in relation to device specific re-interventions, although one late open conversion was required in this group for endograft erosion and infection. Given an anticipated 45% early mortality attending open repair of RDA,[21] a one-year survival of 37% after TEVAR repair appears favorable.

Traumatic aortic tear. Even prior to commercial approval of a TEVAR device, in the US there has been a rapid shift to TEVAR repair among surgeons treating TT.[7,20,24] Since TT patients are typically victims of multisystem trauma, associated injuries are the rule and have greatly impacted treatment results for TT even before the TEVAR era. Recognizing that prompt operation for aortic repair was often prohibited by associated injury, a policy of "sur-

gical delay" evolved wherein TT patients surviving to hospitalizations were managed with anti-impulse therapy to "contain" the aortic lesion until treatment of associated injuries permitted repair of TT.[25,26] The obvious flaw in this strategy was the risk of interval rupture, estimated at 5%-15% in some studies.[26] Despite improvements in overall results with open repair, operative mortality remains significant even in contemporary reports, ranging from 14%-23% in several recent meta-analysis and multicenter registries.[7,20,23,24] Virtually all of these reports detail highly significant reductions in operative mortality in favor of TEVAR for TT. A single death occurred in our study from acute respiratory distress syndrome related to pulmonary contusion, yet the composite end-point of 10% was driven by a paraplegia complication in one of our TT patients. This complication is vanishingly rare after TEVAR treatment of TT; in 2008 publications including two meta-analyses and one multicenter registry, which in composite detail 715 TT patients managed with TEVAR, only two cases of spinal cord ischemic (SCI) complications were detailed.[7,20,24] Indeed, these same three studies compare the risk of SCI with TEVAR vs. open operation for TT and show a statistically significant benefit for TEVAR (0% vs. 5%-7%). In an American Association for the Surgery of Trauma report, a single paraplegia case with TEVAR precluded statistical benefit as paraplegia accompanying open repair was an admirable 2.9%, essentially half the risk of that reported in recent meta-analyses.[20,24] Our patient who sustained paraplegia did have left subclavian artery (LSA) coverage recently identified as a risk factor for SCI after TEVAR.[27] However, given the typical anatomic proximity to the LSA origin, complete coverage thereof (complete coverage occurred in four of 20 subjects, 17 of 20 had complete or partial coverage) will often be required, and in the circumstance of TT, most surgeons would not perform antecedent LSA revascularization. Indeed a systematic review of the available literature indicates that out of 229 cases of TEVAR procedures for TT for which procedure detail was available, 75 (32.7%) included LSA coverage.[6] The atypical nature of our patients' cord injury (T4 level; complete motor/sensory) suggests anomalous spinal cord circulation. A relevant consideration is the "degree of polytrauma" (as evidenced by ISS) in our patients when compared with other reports. The mean ISS in our study of 36.1 ± 19.9 (range, 1-75) is comparable to that observed in a large multi-institutional trial (39.4)[7] and a large meta-analysis (39.8).[20] Durability considerations referable to TEVAR are often discussed given the young ages of affected patients. Our patients' mean age was 51 years old, slightly older than the mean age in other published large reports.[7,24] Yet, the anatomic circumstances of TT with focal lesions in an otherwise normal aorta should preclude considerations of graft migration and component separation which can be seen with conformational changes in large DTA. Demetriades et al reported a 14% endoleak rate after TEVAR but the timing and/or the definition of these were not specified. Most were successfully managed with a second endovascular procedure.[7] Obviously, attachment site endoleaks should be promptly corrected given the nature of the pathology. The anatomic circumstances of TT with reference to arch anatomy in young patients presents as yet unresolved issues with device design. Partial graft collapse as reported elsewhere did occur in two of our patients within 30 days; both were asymptomatic and discovered on follow-up imaging. One was treated with surgical conversion and the second with repeat TEVAR, the latter with a sustained good result. This phenomenon has been related to both graft oversizing and malapposition on the inferior curve of the aortic arch.[28] Adherence to device sizing guidelines is crucial to prevent this complication.

Complicated type B dissection. In consideration of type B dissection, it has long been appreciated that medical therapy produced equivalent results to surgical graft replacement of the aortic entry tear;[29] yet cTBD (ie, those wherein rupture or malperfusion syndromes occur) have greatly increased mortality when compared with cases wherein medical therapy suffices.[30-33] In such circumstances, mortality is increased 3-fold, being 30% in the original IRAD report.[33] "Complication specific" interventions with surgical[34,35] and/or endovascular fenestration procedures[36,37] produced reasonable results, particularly when considered in the context of excessive mortality attending patients with cTBD.[30,33] Dake et al introduced TEVAR as the equivalent of surgical graft replacement of the proximal entry in 1999, citing 80% resolution of malperfusion syndromes and a 16% mortality in a series of 19 patients.[38] Over the past decade, a variety of mostly small, single center reports, attest to the potential efficacy of TEVAR for cTBD.[8,9,39] A recent cumulative review, which considered 942 patients treated from 1997 to 2007, reported a 9% mortality and 2% paraplegia rate; however, the clinical circumstances in which these patients were treated is unclear;[8] registry studies have the same flaw.[5]

In assessing results of TEVAR for cTBD, two fundamental considerations pertain: first, such patients are relatively uncommon. In a recent IRAD study encompassing a decade of experience, only 125 cTDB patients (ie, 125/571 = 22% of type B patients) required intervention in the acute phase.[19] Second, and perhaps more relevant to our patients, is the heterogeneous spectrum of clinical and anatomic findings in cTBD patients. Clearly, early periprocedural results will vary in accordance with the clinical severity of the cTBD; to wit 15/19 (79%) of our patients were treated for rupture and/or malperfusion. The 16% early mortality (two stroke-related) is within the 3%-21% range noted in several single center series[9,39,40] and cumulative reviews.[8]

In consideration of comparison of TEVAR vs. open surgery, early mortality was halved in our patients compared with literature controls. Similar data was reported by the IRAD investigators. Fattori et al reviewed 125 patients who required intervention for cTBD; 59 (47%) were treated with open surgery (56/59 graft replacement of DTA) and 66 (53%) with an endovascular approach (2/3 of these TEVAR, remainder endovascular fenestration). Open

surgery was associated with greater than three-fold increase in mortality (odds ratio [OR] 3.4).[19]

Paraplegia did not occur in our cTBD patients after TEVAR; the reported incidence of this devastating complication in single center series ranges from (0%-15%)[9,39,40] and may be related to the vagaries of intercostal perfusion from true vs. false lumen, and the length of DTA coverage. In the review of Parker et al, paraplegia occurred in 2% of treated patients.[8] The controversy about length of DTA coverage in aTBD is an issue in need of further study.

Despite the evident focus of resolution of immediate life-threatening complications, TEVAR for cTBD has an additional potential benefit, viz. prevention of late aneurysm formation which is known to complicate 40%-50% of all dissections irrespective of initial medical or surgical therapy.[41] Continued patency of the false lumen has repeatedly been associated with such late aneurysm formation.[42] In our study, favorable aortic remodeling was observed, with true lumen expansion, false lumen thrombosis, and reduction in overall aortic diameters.[14] Similar data have been reported by others.[43]

Ruptured degenerative DTA. As the concept that TEVAR compares favorably with open repair in the elective treatment of intact DTA is now supported by comparative trial data,[1,2,6] an intuitively logical extension would be that such benefit would extend in a more emphatic way, to patients with ruptured DTA. The comparative experience with EVAR for ruptured AAA is both more mature and corroborative with this position.[44,45] The results reported herein support this logic. A short term mortality of 15% in our patients is virtually half that reported for open repair from centers of excellence[15,16] and 1/3 that reported in "real world" NIS data.[21] Our favorable early results are also consistent with emerging longitudinal data in large single center series.[46,47] Despite favorable 30-day data in our patients, mortality during the first year after treatment was substantial in this group.

CONCLUSION

Our study confirmed treatment advantages for TEVAR for thoracic aortic catastrophes when compared with extant data on conventional surgical repair. Overall treatment results at one year indicate a low incidence of graft related complications. While device evolution is hardly complete, TEVAR will be the principle initial treatment for the pathologies studied herein.

AUTHOR CONTRIBUTIONS

Conception and design: RPC, JB, VR
Analysis and interpretation: RPC, RSC, JB, MF, AL, CK
Data collection: JC, AL
Writing the article: RPC, RSC, CK
Critical revision of the article: RPC, RSC, JC, JB, MF, AL, VR, CK
Final approval of the article: RPC, RSC, JB, AL, VR, CK
Statistical analysis: RSC
Obtained funding: N/A
Overall responsibility: RPC

REFERENCES

1. Bavaria JE, Appoo JJ, Makaroun MS, Verter J, Yu ZF, Mitchell RS. Endovascular stent grafting versus open surgical repair of descending thoracic aortic aneurysms in low-risk patients: a multicenter comparative trial. J Thorac Cardiovasc Surg 2007;133:369-77.
2. Matsumura JS, Cambria RP, Dake MD, Moore RD, Svensson LG, Snyder S. International controlled clinical trial of thoracic endovascular aneurysm repair with the Zenith TX2 endovascular graft: 1-year results. J Vasc Surg 2008;47:247-57; discussion 57.
3. Fairman RM, Criado F, Farber M, Kwolek C, Mehta M, White R, et al. Pivotal results of the Medtronic Vascular Talent Thoracic Stent Graft System: the VALOR trial. J Vasc Surg 2008;48:546-54.
4. Makaroun MS, Dillavou ED, Kee ST, Sicard G, Chaikof E, Bavaria J, et al. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. J Vasc Surg 2005;41:1-9.
5. Leurs LJ, Bell R, Degrieck Y, Thomas S, Hobo R, Lundbom J. Endovascular treatment of thoracic aortic diseases: combined experience from the EUROSTAR and United Kingdom Thoracic Endograft registries. J Vasc Surg 2004;40:670-9; discussion 9-80.
6. Hoffer EK, Forauer AR, Silas AM, Gemery JM. Endovascular stent-graft or open surgical repair for blunt thoracic aortic trauma: systematic review. J Vasc Interv Radiol 2008;19:1153-64.
7. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study. J Trauma 2008;64:561-70; discussion 70-1.
8. Parker JD, Golledge J. Outcome of endovascular treatment of acute type B aortic dissection. Ann Thorac Surg 2008;86:1707-12.
9. Szeto WY, McGarvey M, Pochettino A, Moser GW, Hoboken A, Cornelius K, et al. Results of a new surgical paradigm: endovascular repair for acute complicated type B aortic dissection. Ann Thorac Surg 2008;86:87-93; discussion 93-4.
10. Doss M, Balzer J, Martens S, Wood JP, Wimmer-Greinecker G, Fieguth HG, et al. Surgical versus endovascular treatment of acute thoracic aortic rupture: a single-center experience. Ann Thorac Surg 2003;76:1465-9; discussion 1469-70.
11. Girardi LN, Krieger KH, Altorki NK, Mack CA, Lee LY, Isom OW. Ruptured descending and thoracoabdominal aortic aneurysms. Ann Thorac Surg 2002;74:1066-70.
12. Bates B. The nervous system. A guide to physical examination and history taking. Philadelphia, PA: J.B. Lippincott Company; 1987.
13. Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, Bernhard VM, et al. Reporting standards for endovascular aortic aneurysm repair. J Vasc Surg 2002;35:1048-60.
14. Conrad MF, Crawford RS, Kwolek CK, Brewster DC, Brady TJ, Cambria RP. Aortic remodeling after endovascular repair of acute complicated type B aortic dissection. J Vasc Surg 2009;50:510-7.
15. Barbato JE, Kim JY, Zenati M, Abu-Hamad G, Rhee RY, Makaroun MS, et al. Contemporary results of open repair of ruptured descending thoracic and thoracoabdominal aortic aneurysms. J Vasc Surg 2007;45:667-76.
16. Crawford ES, Hess KR, Cohen ES, Coselli JS, Safi HJ. Ruptured aneurysm of the descending thoracic and thoracoabdominal aorta. Analysis according to size and treatment. Ann Surg 1991;213:417-25; discussion 425-6.
17. Melissano G, Bertoglio L, Kahlberg A, Baccellieri D, Marrocco-Trischitta MM, Calliari F, et al. Evaluation of a new disease-specific endovascular device for type B aortic dissection. J Thorac Cardiovasc Surg 2008;136:1012-8.
18. Trimarchi S, Nienaber CA, Rampoldi V, Myrmel T, Suzuki T, Bossone E, et al. Role and results of surgery in acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). Circulation 2006;114(1 Suppl):I357-64.
19. Fattori R, Tsai TT, Myrmel T, Evangelista A, Cooper JV, Trimarchi S, et al. Complicated acute type B dissection: is surgery still the best option? A report from the international registry of acute aortic dissection. JACC Cardiovasc Interv 2008;1:395-402.

20. Tang GL, Tehrani HY, Usman A, Katariya K, Otero C, Perez E, et al. Reduced mortality, paraplegia, and stroke with stent graft repair of blunt aortic transections: a modern meta-analysis. J Vasc Surg 2008;47: 671-5.
21. Schermerhorn ML, Giles KA, Hamdan AD, Dalhberg SE, Hagberg R, Pomposelli F. Population-based outcomes of open descending thoracic aortic aneurysm repair. J Vasc Surg 2008;48:821-7.
22. Rigberg DA, McGory ML, Zingmond DS, Maggard MA, Agustin M, Lawrence PF, et al. Thirty-day mortality statistics underestimate the risk of repair of thoracoabdominal aortic aneurysms: a statewide experience. J Vasc Surg 2006;43:217-22; discussion 223.
23. Takagi H, Kawai N, Umemoto T. A meta-analysis of comparative studies of endovascular versus open repair for blunt thoracic aortic injury. J Thorac Cardiovasc Surg 2008;135:1392-4.
24. Xenos ES, Abedi NN, Davenport DL, Minion DJ, Hamdallah O, Sorial EE, et al. Meta-analysis of endovascular vs open repair for traumatic descending thoracic aortic rupture. J Vasc Surg 2008;48:1343-51.
25. Neschis DG, Scalea TM, Flinn WR, Griffith BP. Blunt aortic injury. N Engl J Med 2008;359:1708-16.
26. Akins CW, Buckley MJ, Daggett W, McIlduff JB, Austen WG. Acute traumatic disruption of the thoracic aorta: a ten-year experience. Ann Thorac Surg 1981;31:305-9.
27. Buth J, Harris PL, Hobo R, van Eps R, Cuypers P, Duijm L, et al. Neurologic complications associated with endovascular repair of thoracic aortic pathology: incidence and risk factors. a study from the European Collaborators on Stent/Graft Techniques for Aortic Aneurysm Repair (EUROSTAR) registry. J Vasc Surg 2007;46:1103-10; discussion 1110-1.
28. van Prehn J, van Herwaarden JA, Muhs BE, Arnofsky A, Moll FL, Verhagen HJ. Difficulties with endograft sizing in a patient with traumatic rupture of the thoracic aorta: the possible influence of hypovolemic shock. J Vasc Surg 2008;47:1333-6.
29. Glower DD, Fann JI, Speier RH, Morrison L, White WD, Smith LR, et al. Comparison of medical and surgical therapy for uncomplicated descending aortic dissection. Circulation 1990;82(5 Suppl):IV39-46.
30. Cambria RP, Brewster DC, Gertler J, Moncure AC, Gusberg R, Tilson MD, et al. Vascular complications associated with spontaneous aortic dissection. J Vasc Surg 1988;7:199-209.
31. Bossone E, Rampoldi V, Nienaber CA, Trimarchi S, Ballotta A, Cooper JV, et al. Usefulness of pulse deficit to predict in-hospital complications and mortality in patients with acute type A aortic dissection. Am J Cardiol 2002;89:851-5.
32. Suzuki T, Mehta RH, Ince H, Nagai R, Sakomura Y, Weber F, et al. Clinical profiles and outcomes of acute type B aortic dissection in the current era: lessons from the International Registry of Aortic Dissection (IRAD). Circulation 2003;108 Suppl 1:II312-7.
33. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA 2000;283: 897-903.
34. Elefteriades JA, Lovoulos CJ, Coady MA, Tellides G, Kopf GS, Rizzo JA. Management of descending aortic dissection. Ann Thorac Surg 1999;67:2002-5; discussion 2014-9.
35. Lauterbach SR, Cambria RP, Brewster DC, Gertler JP, Lamuraglia GM, Isselbacher EM, et al. Contemporary management of aortic branch compromise resulting from acute aortic dissection. J Vasc Surg 2001;33:1185-92.
36. Slonim SM, Miller DC, Mitchell RS, Semba CP, Razavi MK, Dake MD. Percutaneous balloon fenestration and stenting for life-threatening ischemic complications in patients with acute aortic dissection. J Thorac Cardiovasc Surg 1999;117:1118-26.
37. Barnes DM, Williams DM, Dasika NL, Patel HJ, Weder AB, Stanley JC, et al. A single-center experience treating renal malperfusion after aortic dissection with central aortic fenestration and renal artery stenting. J Vasc Surg 2008;47:903-10; discussion 910-1.
38. Dake MD, Kato N, Mitchell RS, Semba CP, Razavi MK, Shimono T, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med 1999;340:1546-52.
39. Feezor RJ, Martin TD, Jr. PJH, Beaver TM, Kledell CT, Lee A. Early outcomes after endovascular management of acute, complicated type B aortic dissection. J Vasc Surg 2009;49:561-6; discussion 566-7.
40. Verhoye JP, Miller DC, Sze D, Dake MD, Mitchell RS. Complicated acute type B aortic dissection: midterm results of emergency endovascular stent-grafting. J Thorac Cardiovasc Surg 2008;136:424-30.
41. Hata M, Shiono M, Inoue T, Sezai A, Niino T, Negishi N, et al. Optimal treatment of type B acute aortic dissection: long-term medical follow-up results. Ann Thorac Surg 2003;75:1781-4.
42. Bernard Y, Zimmermann H, Chocron S, Litzler JF, Kastler B, Etievent JP, et al. False lumen patency as a predictor of late outcome in aortic dissection. Am J Cardiol 2001;87:1378-82.
43. Rodriguez JA, Olsen DM, Lucas L, Wheatley G, Ramaiah V, Diethrich EB. Aortic remodeling after endografting of thoracoabdominal aortic dissection. J Vasc Surg 2008;47:1188-94.
44. Moore R, Nutley M, Cina CS, Motamedi M, Faris P, Abuznadah W. Improved survival after introduction of an emergency endovascular therapy protocol for ruptured abdominal aortic aneurysms. J Vasc Surg 2007;45:443-50.
45. Sadat U, Boyle JR, Walsh SR, Tang T, Varty K, Hayes PD. Endovascular vs open repair of acute abdominal aortic aneurysms--a systematic review and meta-analysis. J Vasc Surg 2008;48:227-36.
46. Mehta M, R. Clement Darling I, Paty PSK, Roddy SP, Taggert JB, Sternbach Y, et al. Endovascular vs. open repair of ruptured thoracic aortic aneurysms: mid-term outcomes of a prospective intent-to-treat study. In: Society of Vascular Surgery Annual Meeting; 2008; San Diego, CA; 2008.
47. Patel HJ, Williams DM, Upchurch GR, Deeb M. A 15 year comparative analysis of open and endovascular repair for the ruptured descending thoracic aorta. In: Society for Clinical Vascular Surgery; 2009; Ft. Lauderdale, Fl.; 2009.

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Additional material for this article may be found online at www.jvascsurg.org.

Appendix A (online only). Study centers with individual site enrollment

Site name/principal investigators	*Acute complicated dissection n (%)*	*Traumatic transection n (%)*	*Aneurysm rupture n (%)*	*Total n*
Subjects Enrolled	19	20	20	59
Stanford University Hospital Daniel Sze, MD	0 (0%)	0 (0%)	1 (5%)	2
Arizona Heart Institute Venkatesh Ramaiah, MD	4 (21%)	1 (5%)	1 (5%)	6
Northwestern Memorial Hospital Mark Morash, MD	0 (0%)	0 (0%)	1 (5%)	1
University of Pittsburgh Jae Cho, MD	0 (0%)	2 (10%)	3 (15%)	5
University of Virginia Alan Matsumoto, MD	0 (0%)	1 (5%)	0 (0%)	1
Massachusetts General Hospital Richard P. Cambria, MD	2 (11%)	3 (15%)	4 (20%)	9
Greenville Memorial Hospital Eugene Langan, MD	0 (0%)	3 (15%)	0 (0%)	3
Emory University Hospital Karthikeshwar Kasirajan, MD	0 (0%)	1 (5%)	2 (10%)	3
The Vascular Group, Albany, NY Manish Mehta, MD, MPH	0 (0%)	0 (0%)	1 (5%)	1
University of Florida Anthony Lee, MD	3 (16%)	3 (15%)	0 (0%)	6
University of Pennsylvania Joseph Bavaria, MD	6 (32%)	1 (5%)	3 (15%)	10
Baylor College of Medicine Alan Lumsden, MD	2 (11%)	2 (10%)	0 (0%)	4
University of North Carolina Mark Farber, MD	2 (11%)	0 (0%)	4 (20%)	6
University of South Florida Martin Back, MD	0 (0%)	2 (10%)	0 (0%)	2

APPENDIX B (ONLINE ONLY)

Open controls

1. Doss M, Balzer J, Martens S, Wood JP, Wimmer-Greinecker G, Fieguth HG, Moritz A. Surgical versus endovascular treatment of acute thoracic aortic rupture: a single-center experience. Ann Thorac Surg 2003;76:1465-9; discussion 1469-70.
2. Girardi LN, Krieger KH, Altorki NK, Mack CA, Lee LY, Isom OW. Ruptured descending and thoracoabdominal aortic aneurysms. Ann Thorac Surg 2002;74:1066-70.
3. Ayabe T, Nakamura K, Yano M, Onitsuka T. [Clinical study on the surgical cases of the ruptured thoracic aortic aneurysm]. Kyobu Geka 2003;56:555-9.
4. Fabian TC, Richardson JD, Croce MA, Smith JS Jr, Rodman G Jr, Kearney PA, et al. Prospective study of blunt aortic injury: Multicenter Trial of the American Association for the Surgery of Trauma. J Trauma 1997;42:374-80; discussion 380-3.
5. Fann JI, Smith JA, Miller DC, Mitchell RS, Moore KA, Grunkemeier G, et al. Surgical management of aortic dissection during a 30-year period. Circulation 1995;92(9 Suppl):II113-21.
6. Fradet G, Jamieson WR, Janusz MT, Ling H, Miyagishima RT, Munro AI. Aortic dissection: current expectations and treatment. Experience with 258 patients over 20 years. Can J Surg 1990;33:465-9.
7. Galloway AC, Schwartz DS, Culliford AT, Ribakove GH, Grossi EA, Esposito RA, et al. Selective approach to descending thoracic aortic aneurysm repair: a ten-year experience. Ann Thorac Surg 1996;62:1152-7.
8. Genoni M, von Segesser LK, Carrel T, Baumann PC, Turina M. [Type B aortic dissections: surgical technique and results]. Helv Chir Acta 1994;60:1151-7.
9. Kawachi Y, Toshima Y, Nakashima A, Arinaga K, Komesu I. Emergency surgery results in life-threatening thoracic aortic disease. Jpn J Thorac Cardiovasc Surg 2002;50:158-64.
10. Kwon CC, Gill IS, Fallon WF, Yowler C, Akhrass R, Temes RT, Malangoni MA. Delayed operative intervention in the management of traumatic descending thoracic aortic rupture. Ann Thorac Surg 2002;74:S1888-91; discussion S1892-8.
11. Langanay T, Verhoye JP, Corbineau H, Agnino A, Derieux T, Menestret P, et al. Surgical treatment of acute traumatic rupture of the thoracic aorta a timing reappraisal? Eur J Cardiothorac Surg 2002;21:282-7.

12. Ott MC, Stewart TC, Lawlor DK, Gray DK, Forbes TL. Management of blunt thoracic aortic injuries: endovascular stents versus open repair. J Trauma 2004;56:565-70.
13. Palma JH, Almeida DR, Carvalho AC, Andrade JC, Buffolo E. Surgical treatment of acute type B aortic dissection using an endoprosthesis (elephant trunk). Ann Thorac Surg 1997;63:1081-4.
14. Razzouk AJ, Gundry SR, Wang N, del Rio MJ, Varnell D, Bailey LL. Repair of traumatic aortic rupture: a 25-year experience. Arch Surg 2000;135:913-8; discussion 919.
15. Sasaki S, Yasuda K, Kunihara T, Shiiya N, Murashita T, Matsui Y, Sakuma M. Surgical results of Stanford type B aortic dissection. Comparisons between partial and subtotal replacement of the dissected aorta. J Cardiovasc Surg (Torino) 2000;41:227-32.
16. Sweeney MS, Young DJ, Frazier OH, Adams PR, Kapusta MO, Macris MP. Traumatic aortic transections: eight-year experience with the "clamp-sew" technique. Ann Thorac Surg 1997;64:384-7; discussion 387-9.
17. Symbas PN, Sherman AJ, Silver JM, Symbas JD, Lackey JJ. Traumatic rupture of the aorta: immediate or delayed repair? Ann Surg 2002;235:796-802.

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18. Alric P, Berthet JP, Branchereau P, Veerapen R, Marty-Ane CH. Endovascular repair for acute rupture of the descending thoracic aorta. J Endovasc Ther 2002;9 Suppl 2:II51-9.
19. Herold U, Piotrowski J, Baumgart D, Eggebrecht H, Erbel R, Jakob H. Endoluminal stent graft repair for acute and chronic type B aortic dissection and atherosclerotic aneurysm of the thoracic aorta: an interdisciplinary task. Eur J Cardiothorac Surg 2002;22:891-7.
20. Scharrer-Pamler R, Kotsis T, Kapfer X, Gorich J, Sunder-Plassmann L. Endovascular stent-graft repair of ruptured aortic aneurysms. J Endovasc Ther 2003;10:447-52.
21. Sunder-Plassmann L, Scharrer-Pamler R, Liewald F, Kapfer X, Gorich J, Orend KH. Endovascular exclusion of thoracic aortic aneurysms: mid-term results of elective treatment and in contained rupture. J Card Surg 2003;18:367-74.
22. Bortone AS, Schena S, D'Agostino D, Dialetto G, Paradiso V, Mannatrizio G, et al. Immediate versus delayed endovascular treatment of post-traumatic aortic pseudoaneurysms and type B dissections: retrospective analysis and premises to the upcoming European trial. Circulation 2002;106(12 Suppl 1):I234-40.
23. Dake MD, Kato N, Mitchell RS, Semba CP, Razavi MK, Shimono T, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med 1999;340:1546-52.
24. Grabenwoger M, Fleck T, Czerny M, Hutschala D, Ehrlich M, Schoder M, et al. Endovascular stent graft placement in patients with acute thoracic aortic syndromes. Eur J Cardiothorac Surg 2003;23:788-93; discussion 793.
25. Hutschala D, Fleck T, Czerny M, Ehrlich M, Schoder M, Lammer J, et al. Endoluminal stent-graft placement in patients with acute aortic dissection type B. Eur J Cardiothorac Surg 2002;21:964-9.
26. Slonim SM, Miller DC, Mitchell RS, Semba CP, Razavi MK, Dake MD. Percutaneous balloon fenestration and stenting for life-threatening ischemic complications in patients with acute aortic dissection. J Thorac Cardiovasc Surg 1999;117:1118-26.
27. Chabbert V, Otal P, Bouchard L, Soula P, Van TT, Kos X, et al. Midterm outcomes of thoracic aortic stent-grafts: complications and imaging techniques. J Endovasc Ther 2003;10:494-504.
28. Fattori R, Napoli G, Lovato L, Grazia C, Piva T, Rocchi G, et al. Descending thoracic aortic diseases: stent-graft repair. Radiology 2003;229:176-83.
29. Karmy-Jones R, Hoffer E, Meissner MH, Nicholls S, Mattos M. Endovascular stent grafts and aortic rupture: a case series. J Trauma 2003;55:805-10.
30. Lachat M, Pfammatter T, Witzke H, Bernard E, Wolfensberger U, Künzli A, Turina M. Acute traumatic aortic rupture: early stent-graft repair. Eur J Cardiothorac Surg 2002;21:959-63.
31. Orend KH, Scharrer-Pamler R, Kapfer X, Kotsis T, Gorich J, Sunder-Plassmann L. Endovascular treatment in diseases of the descending thoracic aorta: 6-year results of a single center. J Vasc Surg 2003;37:91-9.

Appendix C (online only)

Time post treatment (days)	*N at risk at start of interval*	*N events during interval**	*N censored during interval**	*% Free from major device related adverse event*	*95% CI*
Group: Acute Complicated Dissection					
0	19	2 (2)	0 (0)	0.895	(0.641, 0.973)
(0-91)	17	2 (4)	2 (2)	0.786	(0.525, 0.914)
(91-182)	13	1 (5)	0 (2)	0.726	(0.459, 0.876)
(182-273)	12	0 (5)	0 (2)	0.726	(0.459, 0.876)
(273-364)	12	0 (5)	0 (2)	0.726	(0.459, 0.876)

Appendix C (online only). Continued

Time post treatment (days)	*N at risk at start of interval*	*N events during interval**	*N censored during interval**	*% Free from major device related adverse event*	*95% CI*
(364-455)	12	0 (5)	4 (6)	0.726	(0.459, 0.876)
(455-546)	8	0 (5)	8 (14)	0.726	(0.459, 0.876)
Group: Aneurysm Rupture					
0	20	0 (0)	0 (0)	1.000	(1.000, 1.000)
(0-91)	20	6 (6)	5 (5)	0.649	(0.371, 0.828)
(91-182)	9	0 (6)	3 (8)	0.649	(0.371, 0.828)
(182-273)	6	0 (6)	1 (9)	0.649	(0.371, 0.828)
(273-364)	5	0 (6)	1 (10)	0.649	(0.371, 0.828)
(364-455)	4	0 (6)	1 (11)	0.649	(0.371, 0.828)
(455-546)	3	0 (6)	3 (14)	0.649	(0.371, 0.828)
Group: Traumatic Transection					
0	20	0 (0)	0 (0)	1.000	(1.000, 1.000)
(0-91)	20	3 (3)	3 (3)	0.835	(0.570, 0.944)
(91-182)	14	0 (3)	1 (4)	0.835	(0.570, 0.944)
(182-273)	13	0 (3)	1 (5)	0.835	(0.570, 0.944)
(273-364)	12	0 (3)	1 (6)	0.835	(0.570, 0.944)
(364-455)	11	0 (3)	3 (9)	0.835	(0.570, 0.944)
(455-546)	8	0 (3)	8 (17)	0.835	(0.570, 0.944)

CI, Confidence interval.
*****Number in parenthesis represents cumulative events or censored observations through end of interval.

Appendix D (online only). Report detailed and/or summarized report

Time post treatment (days)	*N at risk at start of interval*	*N events during interval**	*N censored during interval**	*% Survival*	*95% CI*
Group: Acute Complicated Dissection					
0	19	0 (0)	0 (0)	1.000	(1.000, 1.000)
(0-12)	19	3 (3)	0 (0)	0.842	(0.587, 0.946)
(12-24)	16	0 (3)	0 (0)	0.842	(0.587, 0.946)
(24-36)	16	1 (4)	0 (0)	0.789	(0.532, 0.915)
(36-48)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(48-60)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(60-72)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(72-84)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(84-96)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(96-108)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(108-120)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(120-132)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(132-144)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(144-156)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(156-168)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(168-180)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(180-192)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(192-204)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(204-216)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(216-228)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(228-240)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(240-252)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(252-264)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(264-276)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(276-288)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(288-300)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(300-312)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(312-324)	15	0 (4)	0 (0)	0.789	(0.532, 0.915)
(324-336)	15	0 (4)	1 (1)	0.789	(0.532, 0.915)
(336-348)	14	0 (4)	0 (1)	0.789	(0.532, 0.915)
(348-360)	14	0 (4)	0 (1)	0.789	(0.532, 0.915)
Group: Aneurysm Rupture					
0	20	0 (0)	0 (0)	1.000	(1.000, 1.000)
(0-12)	20	2 (2)	0 (0)	0.900	(0.656, 0.974)

Appendix D (online only). Continued

Time post treatment (days)	*N at risk at start of interval*	*N events during interval**	*N censored during interval**	*% Survival*	*95% CI*
(12-24)	18	1 (3)	0 (0)	0.850	(0.604, 0.949)
(24-36)	17	0 (3)	0 (0)	0.850	(0.604, 0.949)
(36-48)	17	0 (3)	1 (1)	0.850	(0.604, 0.949)
(48-60)	16	0 (3)	1 (2)	0.850	(0.604, 0.949)
(60-72)	15	1 (4)	0 (2)	0.793	(0.537, 0.917)
(72-84)	14	0 (4)	0 (2)	0.793	(0.537, 0.917)
(84-96)	14	0 (4)	0 (2)	0.793	(0.537, 0.917)
(96-108)	14	2 (6)	0 (2)	0.680	(0.420, 0.843)
(108-120)	12	0 (6)	0 (2)	0.680	(0.420, 0.843)
(120-132)	12	1 (7)	0 (2)	0.623	(0.366, 0.801)
(132-144)	11	0 (7)	1 (3)	0.623	(0.366, 0.801)
(144-156)	10	0 (7)	0 (3)	0.623	(0.366, 0.801)
(156-168)	10	1 (8)	0 (3)	0.561	(0.308, 0.753)
(168-180)	9	0 (8)	0 (3)	0.561	(0.308, 0.753)
(180-192)	9	0 (8)	0 (3)	0.561	(0.308, 0.753)
(192-204)	9	0 (8)	0 (3)	0.561	(0.308, 0.753)
(204-216)	9	0 (8)	0 (3)	0.561	(0.308, 0.753)
(216-228)	9	0 (8)	0 (3)	0.561	(0.308, 0.753)
(228-240)	9	0 (8)	0 (3)	0.561	(0.308, 0.753)
(240-252)	9	1 (9)	0 (3)	0.499	(0.254, 0.703)
(252-264)	8	0 (9)	0 (3)	0.499	(0.254, 0.703)
(264-276)	8	0 (9)	0 (3)	0.499	(0.254, 0.703)
(276-288)	8	0 (9)	0 (3)	0.499	(0.254, 0.703)
(288-300)	8	1 (10)	0 (3)	0.436	(0.204, 0.649)
(300-312)	7	0 (10)	0 (3)	0.436	(0.204, 0.649)
(312-324)	7	0 (10)	0 (3)	0.436	(0.204, 0.649)
(324-336)	7	0 (10)	0 (3)	0.436	(0.204, 0.649)
(336-348)	7	0 (10)	0 (3)	0.436	(0.204, 0.649)
(348-360)	7	1 (11)	0 (3)	0.374	(0.158, 0.592)
Group: Traumatic Transection					
0	20	0 (0)	0 (0)	1.000	(1.000, 1.000)
(0-12)	20	1 (1)	0 (0)	0.950	(0.695, 0.993)
(12-24)	19	0 (1)	0 (0)	0.950	(0.695, 0.993)
(24-36)	19	0 (1)	1 (1)	0.950	(0.695, 0.993)
(36-48)	18	0 (1)	0 (1)	0.950	(0.695, 0.993)
(48-60)	18	1 (2)	0 (1)	0.897	(0.648, 0.973)
(60-72)	17	1 (3)	0 (1)	0.844	(0.591, 0.947)
(72-84)	16	0 (3)	0 (1)	0.844	(0.591, 0.947)
(84-96)	16	0 (3)	0 (1)	0.844	(0.591, 0.947)
(96-108)	16	0 (3)	0 (1)	0.844	(0.591, 0.947)
(108-120)	16	0 (3)	0 (1)	0.844	(0.591, 0.947)
(120-132)	16	0 (3)	0 (1)	0.844	(0.591, 0.947)
(132-144)	16	0 (3)	0 (1)	0.844	(0.591, 0.947)
(144-156)	16	0 (3)	0 (1)	0.844	(0.591, 0.947)
(156-168)	16	0 (3)	1 (2)	0.844	(0.591, 0.947)
(168-180)	15	1 (4)	0 (2)	0.788	(0.528, 0.915)
(180-192)	14	0 (4)	0 (2)	0.788	(0.528, 0.915)
(192-204)	14	0 (4)	0 (2)	0.788	(0.528, 0.915)
(204-216)	14	0 (4)	1 (3)	0.788	(0.528, 0.915)
(216-228)	13	0 (4)	0 (3)	0.788	(0.528, 0.915)
(228-240)	13	0 (4)	0 (3)	0.788	(0.528, 0.915)
(240-252)	13	0 (4)	0 (3)	0.788	(0.528, 0.915)
(252-264)	13	0 (4)	0 (3)	0.788	(0.528, 0.915)
(264-276)	13	0 (4)	0 (3)	0.788	(0.528, 0.915)
(276-288)	13	0 (4)	0 (3)	0.788	(0.528, 0.915)
(288-300)	13	0 (4)	0 (3)	0.788	(0.528, 0.915)
(300-312)	13	0 (4)	0 (3)	0.788	(0.528, 0.915)
(312-324)	13	0 (4)	1 (4)	0.788	(0.528, 0.915)
(324-336)	12	0 (4)	0 (4)	0.788	(0.528, 0.915)
(336-348)	12	0 (4)	0 (4)	0.788	(0.528, 0.915)
(348-360)	12	0 (4)	1 (5)	0.788	(0.528, 0.915)

CI, Confidence interval.

*Number in parenthesis represents cumulative events or censored observations through end of interval.